DISSERTATION ON

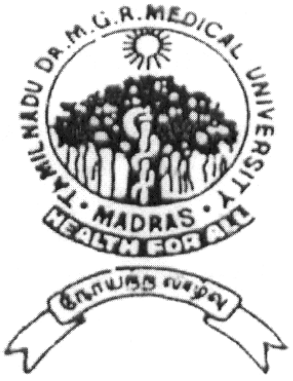
" OCCURRENCE OF SICK EUTHYROID SYNDROME IN ST ELEVATION MYOCARDIAL INFARCTION AND THEIR PROGNOSTIC SIGNIFICANCE"

Submitted in partial fulfilment of

Requirements for

M.D. DEGREE BRANCH I GENERAL MEDICINE

Of
THE TAMILNADU DR.M.G.R. MEDICAL UNIVERSITY, CHENNAI



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CERTIFICATE

This is to certify that this dissertation entitled **"OCCURRENCE OF SICK EUTHYROID SYNDROME IN ST ELEVATION MYOCARDIAL INFARCTION AND THEIR PROGNOSTIC SIGNIFICANCE "** submitted by **Dr. RAMACHANDRAN. P** appearing for Part II M.D. Branch I General Medicine Degree examination in March 2010 is a bonafide record of work done by him under my direct guidance and supervision in partial fulfillment of regulations of the Tamil Nadu Dr. M.G.R. Medical University, Chennai. I forward this to the Tamil Nadu Dr.M.G.R. Medical University, Chennai, Tamil Nadu, India.

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DECLARATION

I solemnly declare that the dissertation titled **"OCCURRENCE OF SICK EUTHYROID SYNDROME IN ST ELEVATION MYOCARDIAL INFARCTION AND THEIR PROGNOSTIC SIGNIFICANCE "** is done by me at Madras Medical College & Govt. General Hospital, Chennai during 2009 under the guidance and supervision of **Prof.A.RADHAKRISHNAN.M.D.**

The dissertation is submitted to The Tamilnadu Dr. M.G.R. Medical University towards the partial fulfillment of requirements for the award of M.D. Degree (Branch I) in General Medicine.

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ABBREVIATIONS

MI	Myocardial Infarction
NTI	Non Thyroidal illness
SES	Sick Euthyroid Syndrome
AMI	Acute Myocardial Infarction
AWMI	Anterior Wall Myocardial Infarction
IWMI	Inferior Wall Myocardial Infarction
LVEF	Left Ventricular Ejection Fraction
T3	Tri iodo thyronine
T4	Thyroxine
TSH	Thyroid Stimulating Hormone
rT3	Reverse Tri iodo thyronine
TRH	Thyrotropin Releasing Hormone
TBPA	Thyroxine Binding Prealbumin
NEFA	Non Esterified Fatty Acid

TBG	Thyroxin Binding Globulin
TNF	Tumor Necrosis Factor
EF	Ejection Fraction
CABG	Coronary Artery Bypass Surgery
GGH	Government General Hospital
ECG	Electrocardiography
ECHO	Echocardiography
HT	Hypertension
DM	Diabetes Mellitus
3,3’,5’ -T3	3,3',5'-triiodothyronine (reverse T_3)
3,5,3’, -T3	3,5,3'-triiodothyronine (T_3)
JVP	Jugular Venous Pressure

CONTENTS

INTRODUCTION

Sick euthyroid syndrome can be described as abnormal findings on thyroid function tests that occur in the setting of a nonthyroidal illness (NTI) without preexisting hypothalamic-pituitary and or thyroid gland dysfunction.[11-13]

A decreased level of serum total triiodothyronine (T_3) is the most common thyroid function abnormality in patients with acute illness[14] and can be detected within 2 hours after the onset of severe physical stress.[15] As the severity of illness progresses, there is gradual development of a more complex syndrome associated with low levels of T_3 and thyroxine (T_4).[16,17]Levels of thyrotropin (TSH) remain unchanged or slightly reduced. The conversion of the pro-hormone (Thyroxine, T4) to the active form is reduced due to decreased 5'-deiodinase activity peripherally and production of reverse T3 (rT3), the inactive metabolite, is increased.

These thyroid hormone changes may be mediated in part by cytokines or other inflammatory mediators, acting at the level of the hypothalamus and pituitary, the thyroid gland, and the hepatic deiodinase system, as well as on binding of thyroxine to thyroid binding globulin.(TBG)

It remains unresolved whether the hormone responses in the sick euthyroid syndrome represent part of an adaptive response, which lowers tissue energy requirements in the face of systemic illness, or a maladaptive response, which induces damaging tissue hypothyroidism.

Consequently, the use of thyroid hormone therapy in the sick euthyroid syndrome is controversial.

Recovery from the underlying illness is accompanied by disappearance of the thyroid abnormalities.

Altered thyroid hormone levels have been reported in starvation,[10] acute and chronic medical illnesses,[29-31] bone marrow transplantation,[8] surgery,[9] trauma,[22],myocardial infarction [4] and, in fact, can be seen in any severe systemic illness.[28]

The more profound the changes in hormone pattern, the poorer the prognosis.[18,19].

Sick euthyroid syndrome has also been demonstrated in acute myocardial infarction and a correlation between the severity of the cardiac damage and the degree of the change in thyroid hormones was postulated.[4]

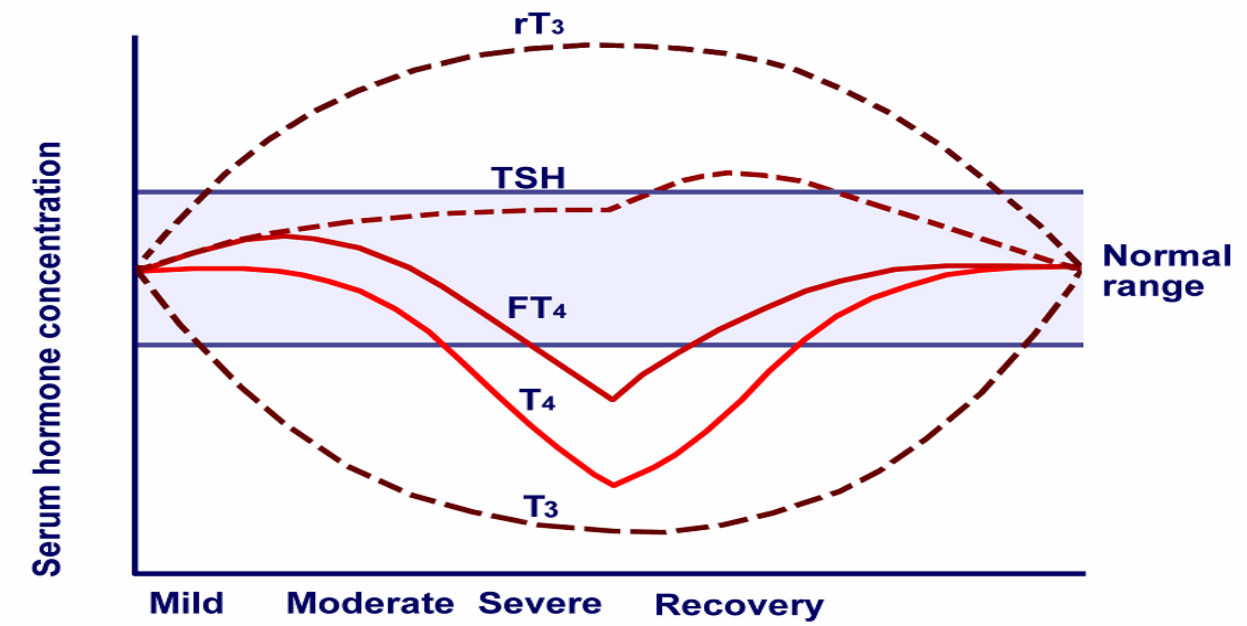
The aim of the present investigation is to study the occurrence of Sick Euthyroid syndrome in patients with ST elevation myocardial infarction and to evaluate whether the presence of sick euthyroid syndrome in these patients have any prognostic significance in determining severity of AMI.

Background

In severe illness of any cause, depression of the thyroid hormone system may occur in otherwise euthyroid patients. In this condition, called the sick euthyroid syndrome, the normal feed-back control of the thyroid homeostasis is changed.[11-13]

Based upon the conviction that patients with systemic illnesses are euthyroid, Wartofsky and Bunnan[14] in 1982 used the term sick euthyroid syndrome to describe the spectrum of thyroid abnormalities associated with nonthyroidal illness.

Relationship between serum thyroid hormone concentrations and severity of Non thyroidal illness (NTI)



In critical illness, correlation between the severity of the illness and the degree of the change in thyroid hormones was described.

Recovery from the underlying illness is accompanied by disappearance of the thyroid abnormalities.

Pituitary-Thyroidal Axis

Secretion of thyroid hormones from the thyroid gland is controlled by pituitary thyrotropin/thyroid-stimulating hormone (TSH) release, which is in turn controlled by hypothalamic thyrotropin-releasing hormone (TRH). Thyroxine and T_3, the main circulating thyroid hormones, exert feedback inhibition at the pituitary and hypothalamic level.[23]

Thyroxine is nearly completely produced in the thyroid gland, and once secreted in the circulation, is peripherally deiodinated to T_3 in the liver, kidney and skeletal muscle .[23] Two peripheral enzymes deiodinate T_4:(1) 5'-monodeiodinase converts T_4 to its active metabolite, 3,5,3'-T_3[23,77]; whereas(2)5-monodeiodinase converts T_4 to reverse T_3 (3,3',5'-T_3), a biologically inactive metabolite. In addition to peripheral deiodination, there is local deiodination of T_4 to T_3 in the pituitary gland.[16,78]

More than 99% of T_4 and T_3 is bound to thyroxine-binding globulin (TBG), T_4-binding prealbumin (TBPA, also termed transthyretin), and albumin. Only free,

unbound T_3 (approximately 0.2% of the total T_3) is metabolically active. Triiodothyronine enters the cell nucleus, binds to its nuclear receptor, and regulates transcription of thyroid hormone-responsive genes, resulting in the physiologic changes associated with thyroid hormone action.[79]

Pathophysiology

Proposed mechanisms explaining abnormalities in thyroid hormone levels

Inhibition of thyroid hormone binding to thyroid-binding proteins and tissues

Some authors propose that serum thyroid hormone abnormalities are due to inhibition of thyroid hormone binding to proteins, thus preventing tests from appropriately reflecting free hormone levels. This binding inhibitor can be present both in the serum and in body tissues and might inhibit uptake of thyroid hormones by cells or prevent binding to nuclear T3 receptors, thus inhibiting the action of the hormone. This inhibitor is associated with the nonesterified fatty acid (NEFA) fraction in the serum. [31,89,51]

Thyroxine-binding globulin decrease and desialation

Thyroxine binding globulin (TBG) is a member of the serine protease inhibitors. Diminished T4 in NTI has been proposed to be due to low TBG caused by protease cleavage at inflammatory sites in acute inflammatory conditions. One other

hypothesis for the cause of disproportionately low serum T4 concentrations in patients with NTI is the presence of abnormal serum binding due to desialation of TBG. [14,29,51]

Cytokines

Cytokines are thought to play a role in NTI—particularly interleukin (IL)-1, IL-6, tumor necrosis factor (TNF)-alpha, and interferon-beta. Cytokines are thought to affect the hypothalamus, the pituitary, or other tissues, inhibiting production of TSH, thyroid-releasing hormone (TRH), thyroglobulin, T3, and thyroid-binding globulins. Cytokines are also thought to decrease the activity of type I deiodinase and to decrease the binding capacity of T3 nuclear receptors. [36,38]

Deiodination

Peripheral deiodination of T4 to T3 is impaired, largely secondary to decreased activity of type I deiodinase enzyme, which deiodinates T4 to T3. Diminished enzyme activity accounts for decreased deiodination of T4 to T3.[23,24]

An alternative explanation is that reduced tissue uptake of T4 secondary to deficiency of cytosolic cofactors (eg, nicotinamide adenine dinucleotide phosphate [NADPH], glutathione) results in decreased substrate for type I deiodinase enzyme. Type I deiodinase is a selenoprotein; because selenium deficiency is common in critically ill patients, some propose that selenium deficiency may

contribute to type I deiodinase malfunction. Cytokines (eg, IL-1 beta, TNF-alpha, interferon-gamma) decrease type I deiodinase messenger RNA (mRNA) in vitro. Type I deiodinase does not exist in the pituitary, where T3 levels are within the reference range, because of enhanced local deiodination. This indicates that an enhancement of intrapituitary T4 to T3 conversion exists due to pituitary-specific and brain-specific type II deiodinase.

Inhibition of thyroid-releasing hormone and thyroid-stimulating hormone secretion

Cytokines, cortisol, and leptin, as well as changes in brain thyroid hormone metabolism, affect inhibition and secretion of TRH and TSH. [8,81,34]

Inhibition of plasma membrane transport of iodothyronines

Serum factors, such as bilirubin, NEFA, furanoic acid, hippuric acid, and indoxyl sulphate, which are present in various NTIs, have been shown to inhibit transport of thyroid hormones.

The effects of nonthyroidal illness

Triiodothyronine and reverse triiodothyronine

In healthy people, 20% of T3 production comes from thyroidal secretion and 80% from peripheral deiodination from T4. In NTI, the thyroidal production of T3 is

normal, but the peripheral production of T3 is decreased. The fractional rate of transport of T3 to tissues is unaltered. Production of T3 is decreased, but its clearance is unchanged. Production of rT3 is unchanged, while its clearance is diminished.

Reduced 5'-deiodinase tissue activity, results in decreased T3 production from T4[8] and reduced breakdown of rT3. The decreased production of T3 during early and late starvation has been explained as either a diminished activity of the enzyme (deiodinase) itself or a deficiency of cytosolic cofactors, such as NADPH or glutathione. Specific deiodinative enzymes, 3 of which have been identified, affect deiodination of iodothyronines. Type I deiodinase is present in the liver, kidney, and thyroid and affects both 5 and 5' deiodination of T3. Type II deiodinase is present in the brain, pituitary, and brown adipose tissue and is active only in 5' deiodination. Type III deiodinase is found particularly in the brain, skin, and placenta, and it deiodinates iodothyronines at the 5 locations. [80,89,90]

type I deiodinase is a selenoprotein and selenium deficiency is common in critically ill patients, selenium deficiency also may contribute to its malfunction.

Cytokines, such as IL-1 beta, TNF-alpha, and interferon-gamma, decrease type I deiodinase mRNA. [59]

Free triiodothyronine

Most studies have found free T3 hormones to be depressed.

Thyroxine

The decrease in the T4 binding of TBG has been used as an explanation for the low plasma T4 concentration in patients with NTI. The existence of a binding inhibitor could explain the observed alterations in T4 and free T4 fraction. TBG levels usually are within the reference range in patients with NTI and are somewhat lower in critically ill patients with low serum T4. Low TBG levels can be explained, according to some proposals, by rapid protease cleavage at inflammatory sites, particularly in acute inflammatory states (in which the decrease in TBG is too rapid to be accounted for by inhibition of synthesis).

In patients with NTI, serum T4 concentration has been demonstrated to be low because much of the circulating TBG in these patients is desialated. In NTI, the fractional rate of T4 transport from serum to tissues is reduced to 50% of the reference range value. This decrement in fractional rate of T4 transport is not related to the serum levels of total or free T4. Because in illness the reduction in the fractional rate of T4 transport from serum to tissues cannot be attributed to alterations in serum T4 binding, consider other causes such as an impairment of transport into tissues. In nonuremic critical illness, it has been demonstrated that

elevated bilirubin or elevated NEFA and low albumin concentration may be at least partially responsible for the T4 transport inhibition in T3-producing tissues (eg, the liver).

A correlation exists between the probability of death and the levels of total T4. When serum T4 levels drop below 4 mcg/dL, the probability of death is about 50%; with serum T4 levels below 2 mcg/dL, the probability of death reaches 80%.[40]

Free thyroxine

No consensus exists as to whether free T4 levels are within the reference range, low, or high. Free T4 is believed to represent the hormone available to tissues. Measurement of total serum T4 has only limited value because nearly all (99.97%) of the circulating T4 is bound to TBG, T4-binding prealbumin (TBPA), and albumin. The rest of the circulating T4 (0.2-0.03%) is free T4. The circulating concentration of these binding proteins is understood to affect the total T4 concentration without necessarily changing the amount of free T4. Usually, TBG levels are within the reference range in patients with NTI and somewhat lower in critically ill patients with low serum T4. Decreased concentrations of one or more of the binding proteins would explain low levels of total T4 but does not explain a significant increase in free T4 fraction, which some patients with NTI exhibit.

Various explanations for the existence of inhibitors of T4 binding have been reported. Although low levels of TBPA and albumin may occur in patients with NTI, even complete inhibition of T4 binding to these proteins has been demonstrated to produce only about a 30% increase in free T4 fraction. Because free T4 fraction is increased above this level in many patients, other factors must be present. The observations of reduced total T4 and free T4 have been explained alternatively as either a fall in TBG levels or an inhibition of thyroid hormone binding to TBG.

Some studies have shown a decrease in the T4 binding of TBG, which has been used as an explanation for the low plasma T4 concentration and, perhaps, the high free T4 fractions, in patients with NTI. Other studies postulate the existence of a binding inhibitor that could explain the observed alterations in free T4 fraction.

The inhibitor also has been demonstrated to interfere with the binding of iodothyronines to solid matrices, thus interfering with the T3 resin uptake and explaining the low FTI found in patients with NTI. The inhibitor appears to be extractable with ether and was associated with the NEFA fraction in the serum. Furthermore, the extracted inhibitor from sera of patients with NTI reduced conversion of T4 to T3 in rat liver homogenates. The inhibitor could be extracted from extra thyroidal tissues as well.

The addition of NEFA to normal serum is able to raise the free T4 fraction only if total NEFA concentration is higher than 3 millimoles in normal serum, representing a NEFA-to-albumin molar ratio greater than 5:1. Because this high NEFA-to-albumin ratio is not reached even in severely ill patients, NEFA is unlikely to influence the circulating free T4 concentration in vivo. Inhibitors of binding were also observed during equilibrium dialysis assay in patients treated with heparin. This is due to an in vitro artifact that is not present in vivo.

Cytokines also can elevate free T4. When TNF-alpha was infused, it was observed that free T4 could elevate transiently in association with a significant rise in free fatty acids. However, other studies question the role of NEFA inhibition or whether any thyroid hormone–binding inhibitor exists at all.

Thyroid hormone receptor expression and DNA binding

In experimental mouse liver models, infection decreased thyroid hormone receptor (TR) expression as well as retinoid X receptor (RXR)–TR DNA binding.

Thyroid-stimulating hormone and thyroid-releasing hormone

Serum TSH is measured with immunometric assays. Serum TSH in NTI typically is within the reference range or reduced. Serum TSH may be markedly low, although it usually not less than 0.05 μIU/ml. These low TSH levels are often observed without significant decrease in T4.

That TSH is not elevated in the presence of low T4 indicates that the patients are not hypothyroid. Diminished release of TRH also is thought perhaps to result in low TSH and, thus, low output of thyroid hormones by the thyroid. Low TRH mRNA in hypothalamic paraventricular nuclei also has been demonstrated. [8,81,34]

Diagnosis of Thyroid Disease in Euthyroid Sick Syndrome

Hypothyroidism

Primary hypothyroidism

Serum level of TSH >30 µU/mL is rarely seen in euthyroid sick syndrome and strongly suggests the diagnosis of primary hypothyroidism. Levels of TSH above 20 µU/mL are found in <3% of patients with nonthyroidal illness [49]

Secondary hypothyroidism

Differentiation between secondary hypothyroidism (pituitary or hypothalamic) and euthyroid sick syndrome may be difficult. Both conditions present with decreased levels of total T_4, T_3, and TSH.

Additional tests, including obtaining basal and/or stimulated cortisol, serum gonadotropin, and prolactin levels may be of help in such cases. If the serum cortisol level is normal or elevated, as would be expected in stressful situations, euthyroid sick syndrome is probably the cause, rather than pituitary dysfunction.

If serum cortisol and gonadotropin levels are low, pituitary dysfunction should be suspected, and treatment with corticosteroids and thyroid hormone supplementation is indicated.

Hyperthyroidism

Hyperthyroid patients who are chronically ill or malnourished may have hypoproteinemia and low levels of TBG that lower their T_4 and T_3 levels. In such patients, an elevated free-T_4 level and a low or undetectable TSH level will confirm the diagnosis of hyperthyroidism.

A previous history of thyroid illness, a history of external radiation, or the presence of goiter and/or a midline neck scar may indicate a primary thyroid condition.

As mentioned before, certain pharmacologic agents may alter the serum concentration of thyroid hormones and should be taken into account in the evaluation of patients with nonthyroidal illness. The concentrations of total T_3, free-T_4, and TSH are reduced in patients treated with dopamine or corticosteroids,[81,45] due to suppression of pituitary TSH release[62] and/or inhibition of conversion of T_4 to T_3.[63] Levels of total and free-T_4 may be increased in patients treated with amiodarone[64] or iodinated radiocontrast agents.[15,65] Intravenous or subcutaneous heparin therapy may result in increased free-T_4

levels,[66] due to in vitro interference with the laboratory assay; however, most such patients remain clinically euthyroid and have normal total T_4 and TSH levels.

As a general rule, it is not prudent to rely solely on a single thyroid test in the evaluation of thyroid function of patients with critical illness. In such patients, a careful assessment of multiple tests may be needed to distinguish patients with euthyroid sick syndrome.[14] In many instances, it is reasonable to delay the final diagnosis for several days to weeks, or after recovery from the acute illness, to determine the correct thyroid status.

Treatment

With recovery from underlying illness, the spectrum of thyroid abnormalities observed in patients with euthyroid sick syndrome rapidly disappears. In critically ill patients, however, the presence of reduced T_3 and T_4 concentrations is associated with increased severity of illness and mortality rate.[8,40] The mortality rate in patients admitted to an intensive care unit is directly correlated with serum T_4 concentration.[40]

Because of the increased incidence of mortality observed in patients with severe illness and low T_4 values, several interventional trials have examined the effect of thyroid hormone supplementation in patients with nonthyroidal illness.

The administration of T_4 (levothyroxine) to critically ill adults has failed to demonstrate a reduction in the mortality rate.[41] Administration of T_4 to neonates with respiratory distress syndrome resulted in no differences in mortality rate or number of days on a ventilator; similarly, T_4 supplementation to preterm neonates showed no differences in neurodevelopment or weight.[67] The lack of beneficial effect with T_4 treatment may be due to the inability of these patients to convert administered T_4 to metabolically active T_3.

Several controlled studies in which T_3 was administered to adult and pediatric patients have provided equivocal results. There are few randomized controlled studies reporting the benefits of T_3 replacement.[68,69] In patients undergoing coronary bypass procedures, T_3 administration improved cardiac output and decreased systemic vascular resistance.[68,69] In a different study, the administration of T_3 at release of cross-clamp after myocardial revascularization resulted in improvement in cardiac indexes and decreased inotropic requirements, postoperative ischemia, mortality rate, and length of hospital stay.[70] Other investigators have examined the effect of T_3 administration on pulmonary function in sepsis. These studies showed improvement in respiratory drive, pulmonary histologic integrity, and surfactant availability.[71]

In contrast, other studies have shown little or no benefit from short-term administration of T_3. In a large, prospective, randomized, double-blind, placebo-controlled trial, T_3 was administered to 211 adults who underwent myocardial revascularization or valve replacement; no benefit could be demonstrated in inotropic requirements or need for mechanical support. Adverse events were the same, despite T_3 administration. Similar studies of T_3 administration to the same spectrum of patients also failed to demonstrate differences in clinical outcome.[72,73]

In the presence of these conflicting results, we do not recommend routine administration of thyroid hormones to patients with euthyroid sick syndrome. Even though no harm has been reported with the administration of T_3 to critically ill patients, evidence does not support the use of thyroid hormone supplements to correct thyroid hormone abnormalities.

In critically ill patients with documented hypothyroidism, thyroid hormone administration is indicated. If therapy is to be given, it cannot be with T_4 alone, because this may fail to promptly increase T_3 levels (due to impaired T_4-to-T_3 conversion). In patients with a variety of underlying illnesses admitted to a medical intensive care unit, intravenous T_4 sufficient to raise the total T_4 and free T_4 to normal levels failed to increase T_3 concentrations, while reverse-T_3 levels did rise with treatment.[41]

Therefore, to restore T_4 and T_3 concentrations to normal during critical illness, patients should be treated with a combination of T_4 and T_3. The recommended replacement dose of T_3 is 50 mg/day, given in divided doses. During treatment, frequent monitoring (every 48 hours) of levels of total and free T_4 and T_3 has been recommended,[32] and dosages should be adjusted to achieve a low-normal serum T_3 level.

Euthyroid sick syndrome and myocardial infarction

In severe illnesses of non thyroidal origin (11–12), including myocardial infarction (1,76) and chronic heart failure (93), down regulation of the thyroid hormone system may occur.

Thyroid hormones have substantial effects on the cardiovascular system (18,94,95,96). The active hormone increases heart rate, contractility, cardiac output, and consumption of oxygen and nutrients, mimicking the effects of catecholamines. However, it also decreases systemic vascular resistance and improves diastolic relaxation, leading to a more efficient use of energy and nutrients.

Whether the euthyroid sick syndrome constitutes an adaptive metabolic response to conserve energy in cardiac disease, or whether it aggravates a patient’s condition, is a matter of debate.

Killip class and myocardial infarction

The Killip classification is a system used in individuals with an acute myocardial infarction (heart attack), in order to risk stratify them. Individuals with a low Killip class are less likely to die within the first 30 days after their myocardial infarction than individuals with a high Killip class.[2]

Killip class I includes individuals with no clinical signs of heart failure

Killip class II includes individuals with rales in the lungs, an S_3 , and elevated JVP.

Killip class III describes individuals with frank acute pulmonary edema.

Killip class IV describes individuals in cardiogenic shock or hypotension (measured as systolic blood pressure lower than 90 mmHg), and evidence of peripheral vasoconstriction (oliguria, cyanosis or sweating).

In Killip class I: mortality rate was found to be at 6%, Killip class II: mortality rate was found to be at 17%, Killip class III: mortality rate was found to be at 38% and Killip class IV: Mortality rate was found to be at 81%.

Ejection Fraction and Myocardial infarction

After acute myocardial infarction presence of low left ventricular ejection fraction are strongly associated with morbidity and mortality. [7]

AIMS & OBJECTIVES

1. To find out the occurrence of sick euthyroid syndrome in acute ST elevation myocardial infarction patients admitted to the coronary care unit at the Govt General Hospital Chennai.

2. To find out the prognostic significance of sick euthyroid syndrome positivity in acute ST elevation myocardial infarction patients admitted to the coronary care unit at the Govt General Hospital Chennai.

MATERIALS & METHODS

SETTING

This study was conducted in the coronary care unit, GGH, Chennai in collaboration of institute of internal medicine, institute of Cardiology and institute of biochemistry. It was a prospective study done during the period from jan 2009 – oct 2009 .50 patients with history, clinical features suggestive of ST elevation myocardial infarction were selected irrespective of age and sex.

ETHICAL APPROVAL

Obtained

STUDY DURATION

This study was conducted for a period of eight months from 1st January 2009 to October 30, 2009.

STUDY DESIGN

To find out the occurrence of sick euthyroid syndrome in acute ST elevation myocardial infarction patients and prognostic significance of sick euthyroid syndrome positivity in acute ST elevation myocardial infarction patients ; a single centre prospective cohort study.

INCLUSION CRITERIA

1. Acute ST elevation myocardial infarction patients admitted to the coronary care unit.

EXCLUSION CRITERIA

1. Patients with past or present history of thyroid dysfunction.

2. Patients taking drugs that will affect thyroid function.

3. Patients with chronic renal failure.

4. Patients with decompensated liver disease.

5. Patients with thyroid function test suggestive of primary Hypothyroidism and Hyperthyroidism.

LABORATORY METHODS

All patients underwent CBC, Blood sugar, Serum Na, Urea, Creatinine, LFT, Fasting lipid profile, Thyroid function tests, ECG and ECHO. The serum of patients was analyzed for thyroid function tests at day 1 (totalT3, total T4, TSH) and in sick euthyroid positive patients repeat thyroid function tests done at day 7 to confirm the reversal of hormone status.

The thyroid function test was done by ELISA method. All samples were obtained before reperfusion therapy. Echocardiography was done during hospital stay.

METHODS

STUDY POPULATION

A total of 65 patients were enrolled for the study from the patients admitted to coronary care unit Govt General Hospital from the period 1st January 2009 to October 31st 2009.

15 patients were excluded as per exclusion criteria. The remaining 50 patients, who satisfied all the inclusion criteria were selected for the study and followed for one week. Written consent was obtained from all patients participating in the study.

All patients were subjected to a detailed clinical history and thorough physical examination as per proforma. The complicated cases identified by KILLIP classification criteria and ejection fraction.

The serum of patients was analyzed for thyroid function tests at day 1 (T3,T4,TSH)and in sick euthyroid positive patients repeat thyroid function tests done at day 7 to confirm the reversal of hormone status.

Day 1samples were obtained before reperfusion therapy.

Echocardiography was done during hospital stay.

All patients underwent CBC, Blood sugar, Serum Na, Urea, Creatinine, LFT, Fasting lipid profile, Thyroid function tests, ECG, ECHO.

FINANCIAL SUPPORT: nil.

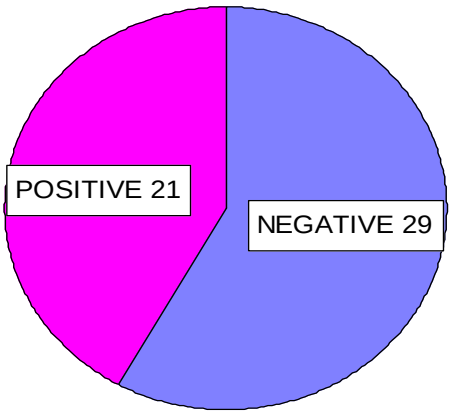
CONFLICT OF INTEREST: nil.

STATISTICAL ANALYSIS

Data analysis was done with use of SPSS, version 10. Descriptive statistics were used to calculate the frequency, mean, median, and standard deviation. To examine the linear trend of the proportions, trend chi-square was used and to find the test of association chi-square was computed.

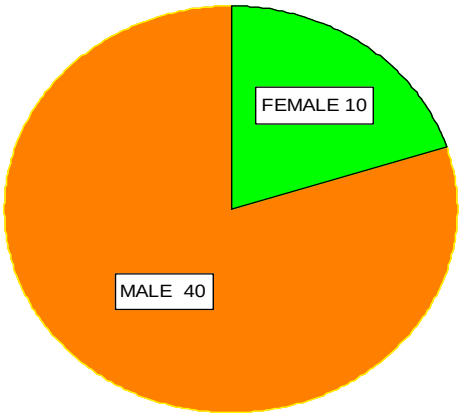
RESULTS

OCCURRENCE OF SICK EUTHYROID AMONG MI PATIENTS



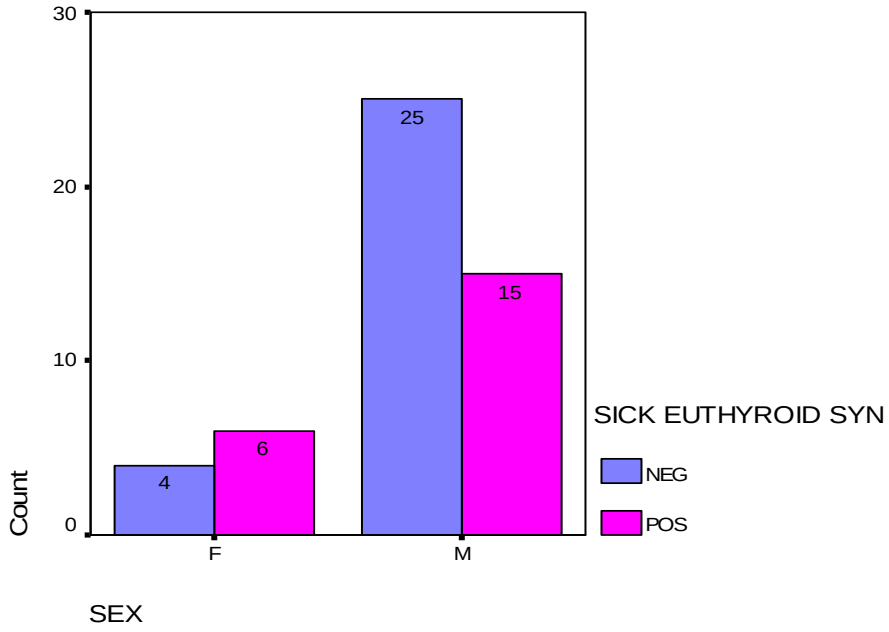
In our study 42% of ST elevation MI patients had sick euthyroid syndrome.so occurrence of sick euthyroid syndrome in our patients 42%.p value was 0.258.

SEX DISTRIBUTION AMONG MI PATIENTS



In our study male to female ratio 4:1

SEX DISTRIBUTION & SICK EUTHYROID SYNDROME

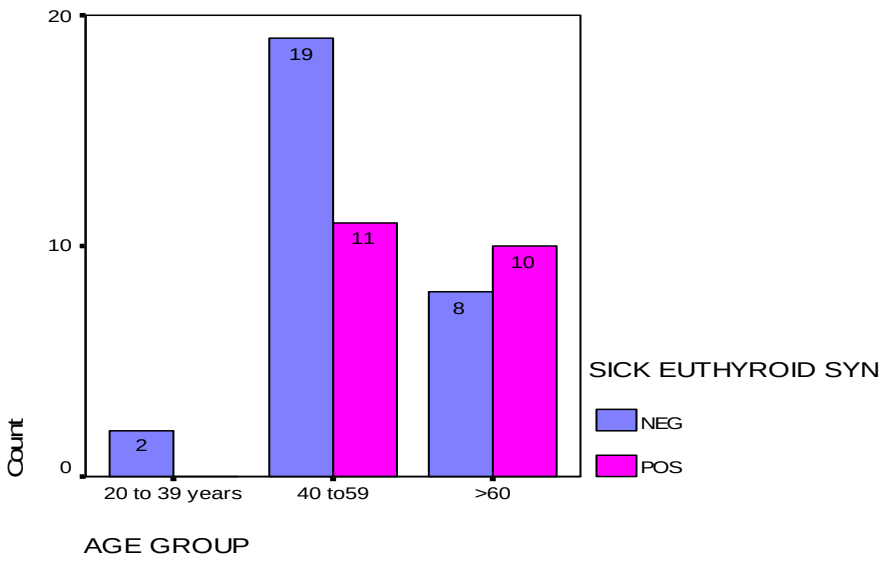


SEX DISTRIBUTION AND SICK EUTHYROID SYNDROME(SES)

	SES +ve	PERCENTAGE	SES-ve	PERCENTAGE	TOTAL
FEMALE	6	60%	4	40%	10
MALE	15	37.5%	25	62.5%	40

In our study occurrence of sick euthyroid syndrome was 60% among females and 37.5% among males. This observation was statistically insignificant with the p value of 0.50.

AGE GROUP & SICK EUTHYROID SYNDROME

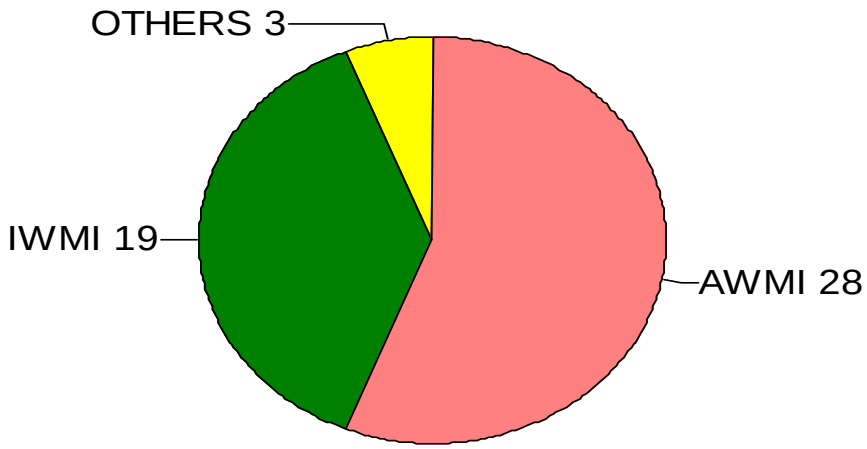


AGE DISTRIBUTION & SICK EUTHYROID SYNDROME

AGE GROUP	SES+ve	PERCENTAGE	SES-ve	PERCENTAGE	TOTAL
20 to 39 years	0	-	2	100%	2
40 to 59 years	11	37%	19	63%	30
>60 years	10	55%	8	45%	18
	21		29		50

In our study in the age group of 20 to 39, no one had sick euthyroid syndrome. In the age group of 40 to 59, 37% had sick euthyroid syndrome. In the age group of >60, 55% of patients had sick euthyroid syndrome. p value was 0.206 that statistically insignificant.

TYPE OF MYOCARDIAL INFARCTION AMONG PATIENTS



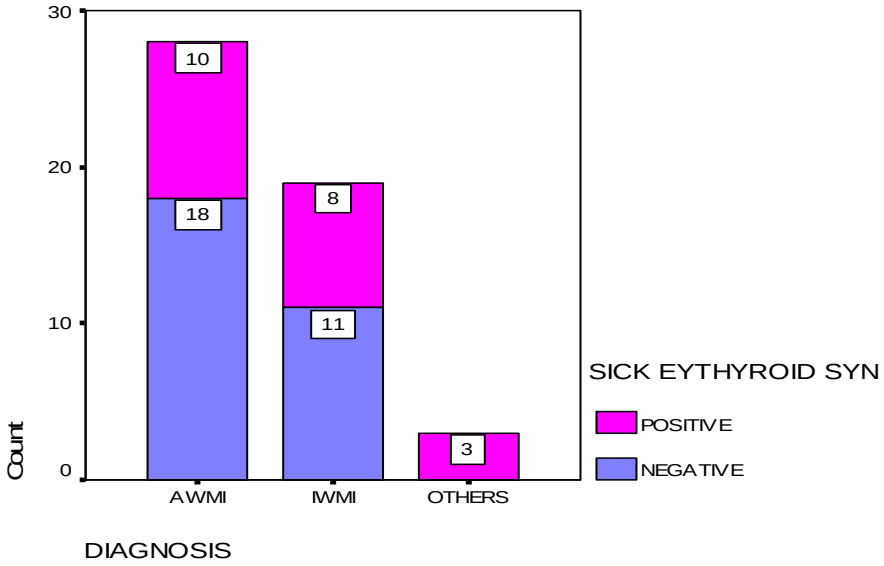
In our study total fifty acute ST elevation MI patients

AWMI patients 28(56%)

IWMI patients 19(38 %)

Others 3(6%)

OCCURRENCE OF SICK EUTHYROID IN DIFFERENT TYPES OF MI

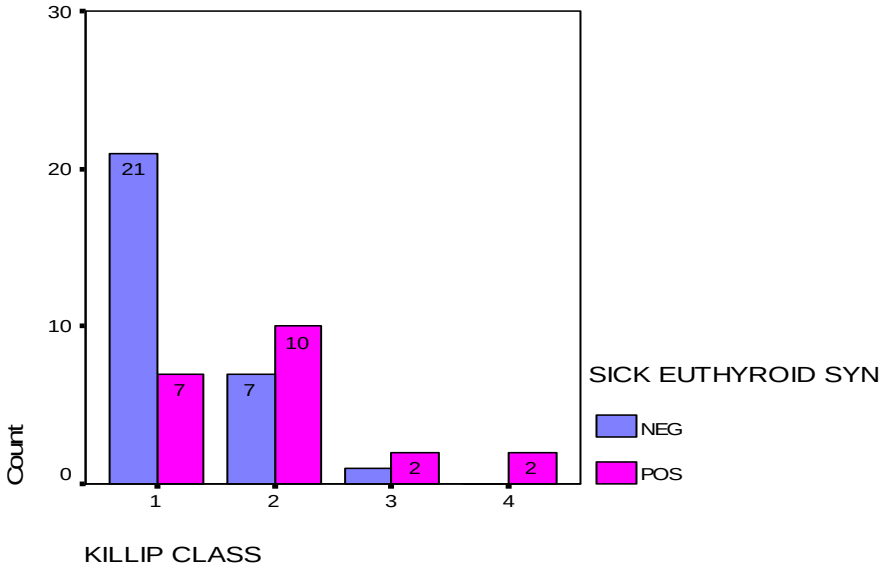


TYPES OF MI AND SICK EUTHYROID SYNDROME

	SES +ve	PERCENTAGE	SES-ve	PERCENTAGE	TOTAL
AWMI	10	36%	18	64%	28
IWMI	8	42%	11	58%	19
OTHERS	3	100%	-	-	3

In our study occurrence of sick euthyroid syndrome in AWMI patients was 36%, in IWMI patients was 42% and in others 100% .p value was 0.20 that statistically insignificant.

OCCURRENCE OF SICK EUTHYROID IN VARIOUS CLASS OF KILLIP



KILLIP CLASS AND SICK EUTHYROID SYNDROME

	SES +ve	PERCENTAGE	SES-ve	PEERCENAGE	TOTAL
KILLIP 1	7	25%	21	75%	28
KILLIP 2	10	59%	7	41%	17
KILLIP 3	2	67%	1	33%	3
KILLIP 4	2	100%	-	-	2
	21		29		50

In our study patients who presented with KILLIP 1 the sick euthyroid syndrome occurrence was 25%, in KILLIP 2 was 59%, in KILLIP 3 was 67% and KILLIP 4 was 100%.P value was 0.05* that statistically significant between the KILLIP 1 and KILLIP 2.

SICK EUTHYROID SYNDROME AND KILLIP CLASS

	KILLIP CLASS								
	I		**II**		**III**		**IV**		**TOTAL**
	No	%	No	%	No	%	No	%	
SES+ve	7	33.3%	10	**47.6%**	2	9.5%	2	9.5%	21
SES-ve	21	**72.4%**	7	**24.1%**	1	3.4%	0		29
TOTAL	28	56%	17	34%	3	6%	2	4%	50

In our study 47.6% of sick euthyroid positive patients are in KILLIP 2 .But sick euthyroid negative patients only 24.1% in KILLIP 2. This observation was statistically significant with p value of 0.032** . Sick euthyroid negative patients are mostly in KILLIP 1.(72.4%)

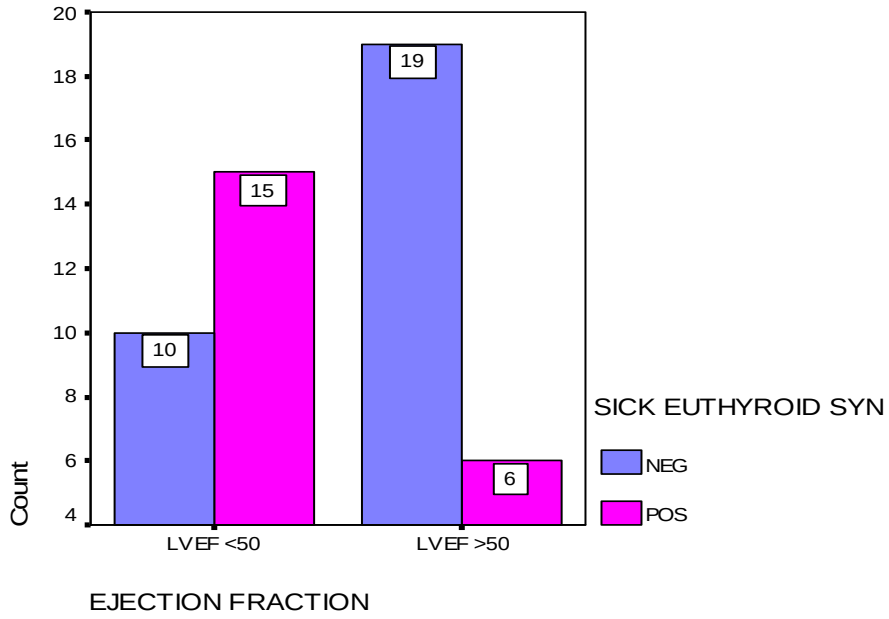
KILLIP CLASS AND MEAN LEVEL OF HORMONES

	MeanT3level ng/ml		**Mean T4 level ng/ml**		**Mean TSH level**	
	ng/ml	Std devi	ng/ml	Std devi	Mic unit/ml	Std devi
KILLIP 1	0.91(28)	.43302	90(28)	17.5778	1.71(28)	1.19447
KILLIP 2	0.63(17)	.31924	70(17)	20.8148	1.46(17)	0.70425
KILLIP 3	0.46(3)	.08145	60(3)	12.490	1.43(3)	0.75719
KILLIP 4	0.39(2)	.04243	52(2)	28.282	.60(2)	0.42426
p value	0.031**		0.001**		0.474	

In our study between the groups of KILLIP class decrease of Mean T3 level, Mean T4 level was statistically significant. But decrease of Mean TSH level was statistically insignificant.

OCCURRENCE OF SICK EUTHYROID SYNDROME

IN LVEF <50 AND LVEF >50



LVEF AND SICK EUTHYROID SYNDROME

	SES+ve	PERCENTAGE	SES-ve	PERCENTAGE	TOTAL
LVEF<50	15	60%	10	40%	25
LVEF>50	6	24%	19	76%	25
	21		29		50

In our study patients who presented with LVEF<50 the occurrence of sick euthyroid syndrome was 60% and patients who presented with LVEF>50 was 24%. p value was 0.01** that statistically significant.

SICK EUTHYROID SYNDROME AND LVEF

	EJECTION FRACTION			
	LVEF<50		**LVEF>50**	
	PATIENTS	**PERCENTAGE**	**PATIENTS**	**PERCENTAGE**
SES+ve	15	71.4%	6	28.6%
SES-ve	10	34.5%	19	65.5%
TOTAL	25	50%	25	50%

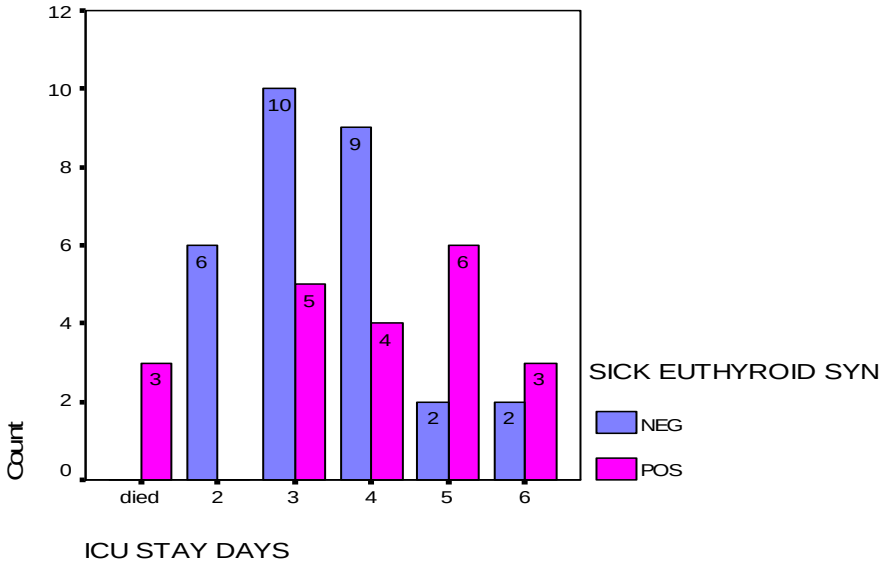
In our study 71.4% of sick euthyroid positive patients are in LVEF<50 But 34.5% of sick euthyroid negative patients only in this group. This observation was statistically significant with p value of 0.010**

LVEF AND MEAN LEVEL OF HORMONES

	MeanT3level ng/ml		**Mean T4 level ng/ml**		**Mean TSH level**	
	ng/ml(n)	Std devi	ng/ml(n)	Std devi	ng/ml(n)	Std devi
LVEF<50	0.62(25)	0.3323	68(25)	20.3046	1.16(25)	0.6305
LVEF>50	0.91	0.4280	92(25)	16.5582	1.97(25)	1.1670
P value	0.009**		0.000**		0.004**	

In our study the decrease of MeanT3level, Mean T4 level and Mean TSH level between the two groups of patients who presented with LVEF <50 and LVEF>50 was observed. This observation was statistically significant.

ICU STAY AND SICK EUTHYROID SYNDROME

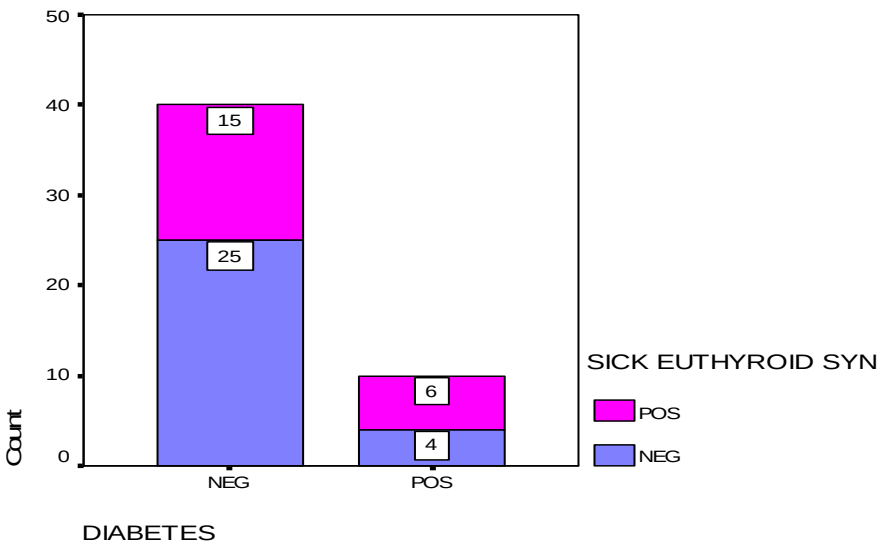


ICU STAY AND SICK EUTHYROID SYNDROME

Sick Euthyroid Syndrome	Mean days	no	Std. Deviation
NEG	3.4483	29	1.12078
POS	4.3889	18	1.09216
Total	3.8085	47	1.19124

In our study in sick euthyroid positive patients mean ICU stay duration was 4.39 days compare to sick euthyroid syndrome negative patients in whom mean ICU stay duration was 3.45 days with p value of 0.007** so this observation was statistically significant.

DIABETES AND SICK EUTHYROID SYNDROME

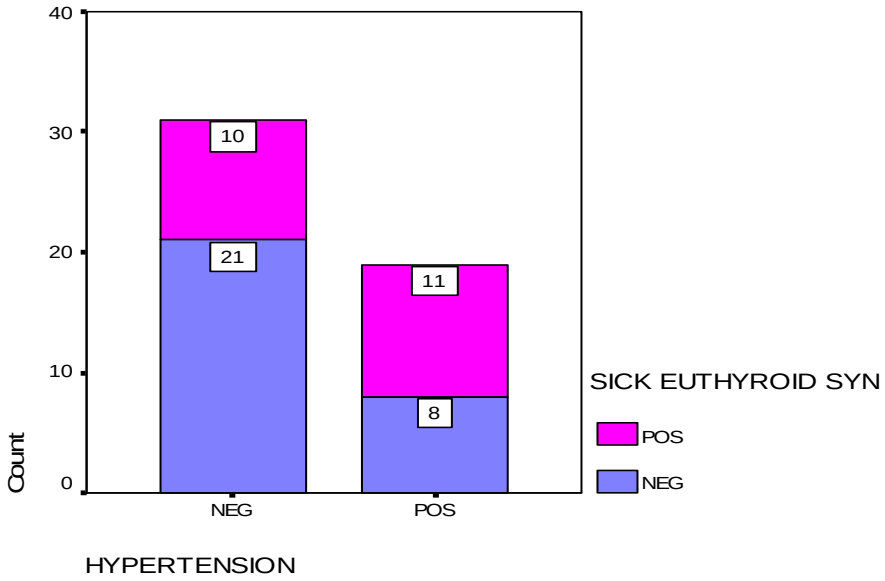


DIABETES AND SICK EUTHYROID SYNDROME

DIABETES	SES+ve	Percentage	SES –ve	Percentage	TOTAL
Present	6	60%	4	40%	10
Absent	15	37.5%	25	62.5%	40
	21		29		50

In our study if the patient who had DIABETES, the occurrence of sick euthyroid syndrome was 60% and patients who presented with NO DIABETES, the occurrence of sick euthyroid syndrome was 37.5%. p value was 0.197 that statistically insignificant.

HYPERTENSION AND SICK EUTHYROID SYNDROME

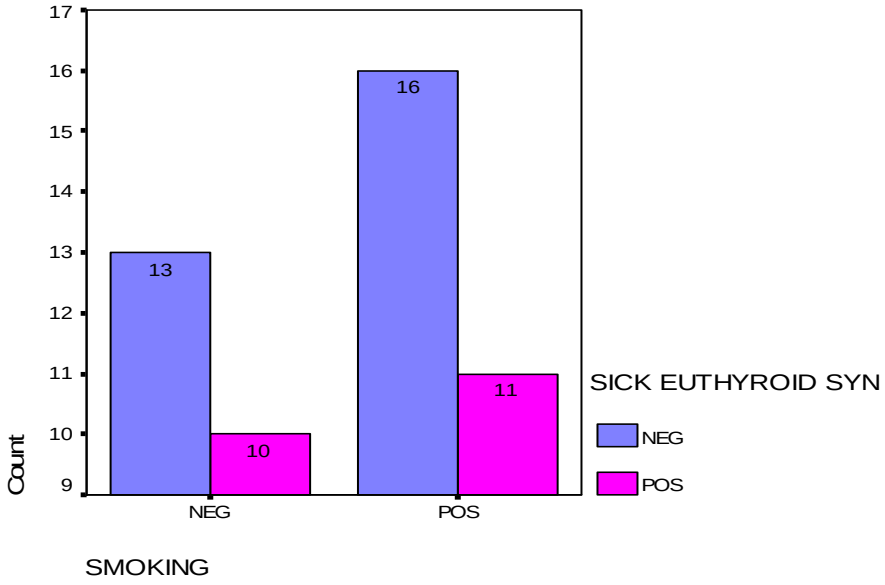


HYPERTENSION AND SICK EUTHYROID SYNDROME

HYPERTENSION	SES +ve	Percentage	SES – ve	Percentage	TOTAL
Present	11	58%	8	42%	19
Absent	10	32%	21	68%	31
	21		29		50

In our study patients who had HYPERTENSION the occurrence of sick euthyroid syndrome was 58% and patients who presented with NO HYPERTENSION the occurrence of sick euthyroid syndrome was 32%. p value was 0.10 that statistically insignificant.

SMOKING AND SICK EUTHYROID SYNDROME

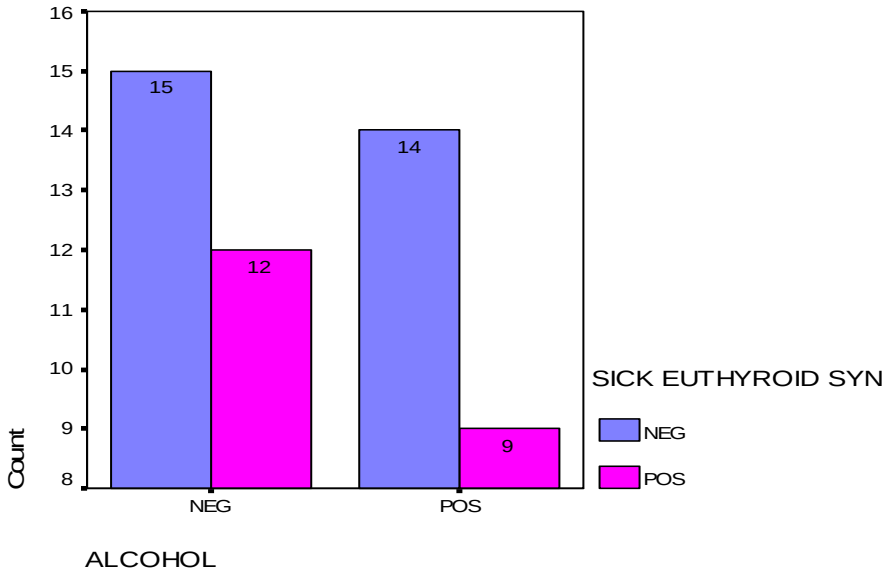


SMOKING AND SICK EUTHYROID SYNDROME

SMOKING	SES+ve	Percentage	SES-ve	Percentage	TOTAL
Present	11	41%	16	59%	27
Absent	10	43%	13	57%	23
	21		29		50

In our study patient who had SMOKING as a risk factor, the occurrence of sick euthyroid syndrome was 41% and patients with NO SMOKING the occurrence of sick euthyroid syndrome was 43% .p value was 0.50 that statistically insignificant.

ALCOHOL INTAKE AND SICK EUTHYROID SYNDROME

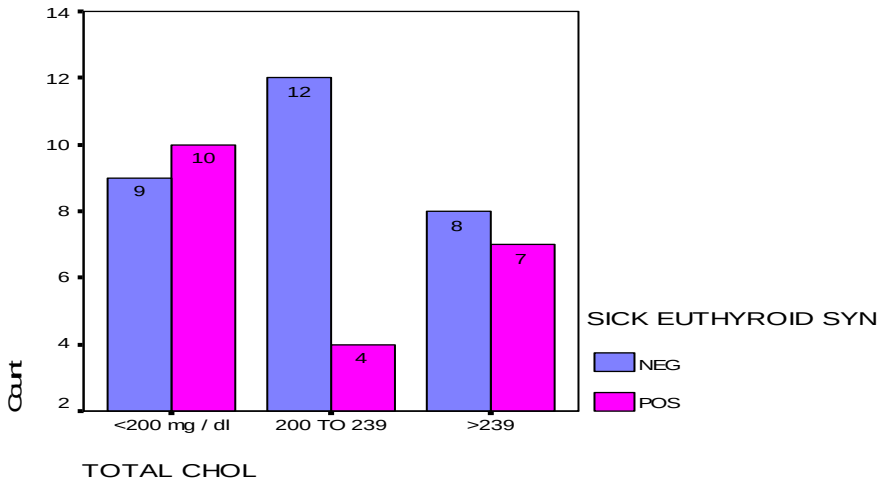


ALCOHOL INTAKE AND SICK EUTHYROID SYNDROME

ALCOHOL	SES+ve	Percentage	SES-ve	Percentage	TOTAL
Present	9	39%	14	61%	23
Absent	12	44%	15	56%	27
	21		29		50

In our study patients who had ALCOHOL intake the occurrence of sick euthyroid syndrome was 39% and patients who presented with no H/O of ALCOHOL intake the occurrence of sick euthyroid syndrome was 44%. This observation was statistically insignificant with p value of 0.50 .

TOTAL CHOLESTEROL LEVEL AND SICK EUTHYROID SYNDROME

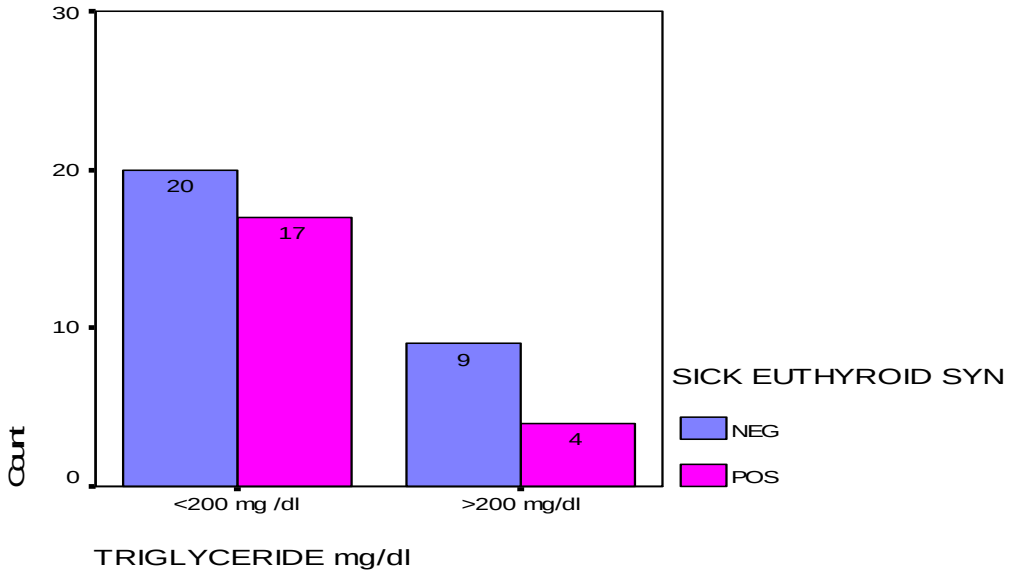


TOTAL CHOLESTEROL LEVEL AND SICK EUTHYROID SYNDROME

	SES +ve	PERCENTAGE	SES-ve	PERCENTAGE	TOTAL
<200 mg	10	53%	9	47%	19
200 to 239 mg	4	25%	12	75%	16
>240 mg	7	47%	8	53%	15
	21		29		50

In our study patients who presented with fasting cholesterol level <200 mg the occurrence of sick euthyroid syndrome was 53%, fasting cholesterol level 200 to 239mg the occurrence of sick euthyroid syndrome was 25% and fasting cholesterol level >240mg the occurrence of sick euthyroid syndrome was 47%. This observation was statistically insignificant with P value of 0.50 .

TRIGLYCERIDE LEVEL AND SICK EUTHYROID SYNDROME

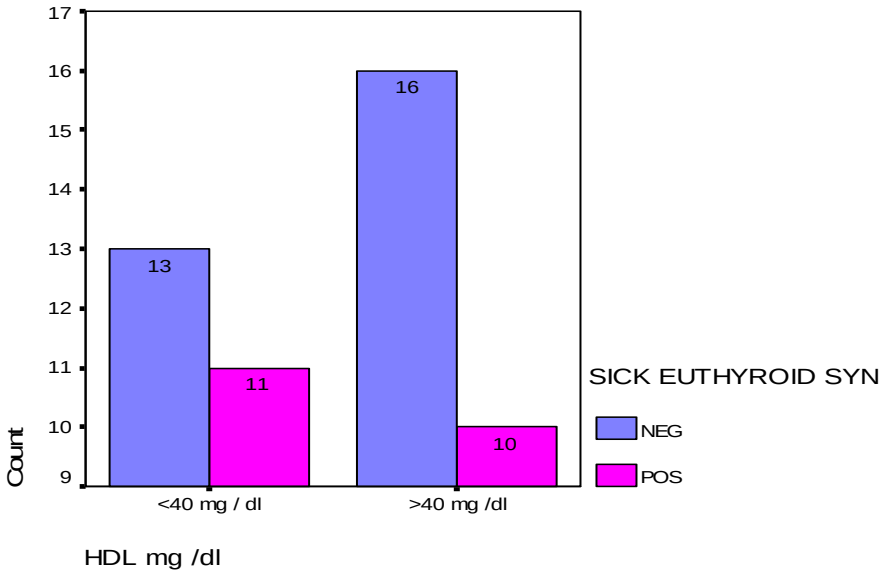


TRIGLYCERIDE LEVEL AND SICK EUTHYROID SYNDROME

TRIGLYCERIDE	SES+ve	PERCENTAGE	SES-ve	PERCENTAGE	TOTAL
<200 mg	17	46%	20	54%	37
>200 mg	4	31%	9	69%	13
	21		29		50

In our study patients who presented with fasting TRIGLYCERIDE level <200 mg the occurrence of sick euthyroid syndrome was 46% and fasting TRIGLYCERIDE level >200mg the occurrence of sick euthyroid syndrome was 31%. This observation was statistically insignificant with P value of 0.50 .

HDL LEVEL AND SICK EUTHYROID SYNDROME

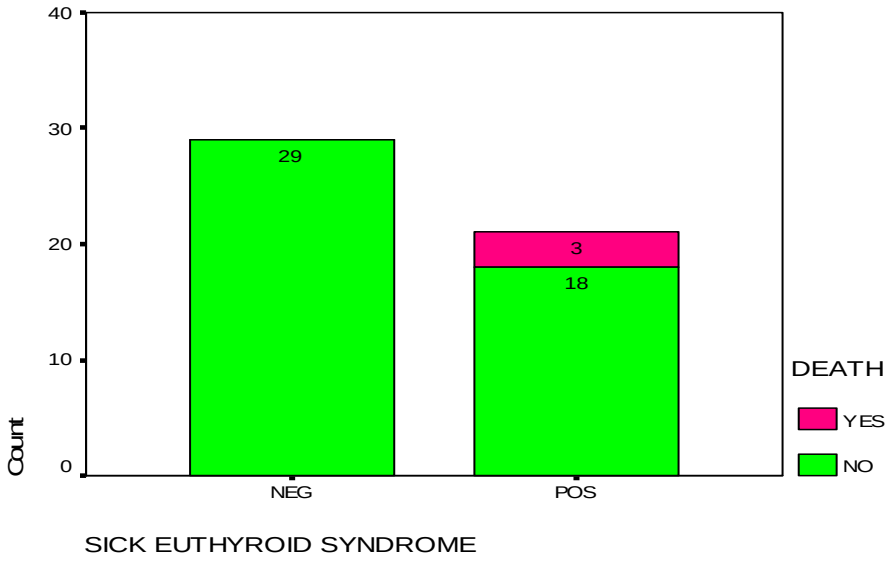


HDL LEVEL AND SICK EUTHYROID SYNDROME

HDL	SES +ve	PERCENTAGE	SES-ve	PERCENTAGE	TOTAL
<40 mg	11	46%	13	54%	24
>40 mg	10	38%	16	62%	26
	21		29		50

In our study patients who presented with fasting HDL level <40mg the occurrence of sick euthyroid syndrome was 46% and fasting HDL level >40mg the occurrence of sick euthyroid syndrome was 38%. This observation was statistically insignificant with P value of 0.50 .

DEATH AND SICK EUTHYROID SYNDROME



	SES +ve	PERCENTAGE	SES-ve	PERCENTAGE	
MALE	2	100%	-	-	2
FEMALE	1	100%	-	-	1

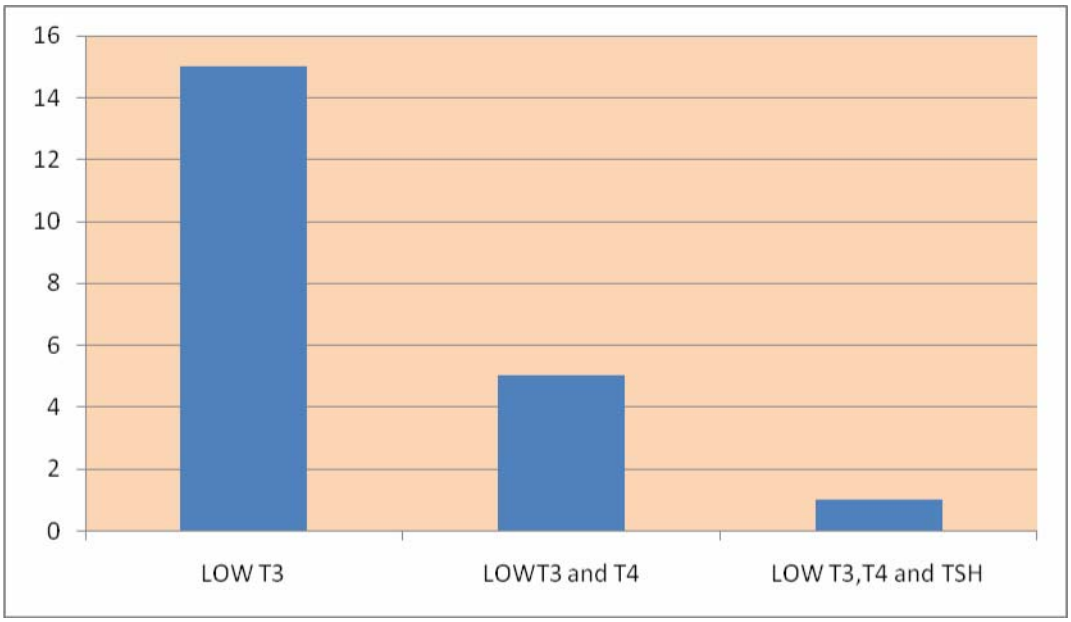
In our study occurrence of sick euthyroid syndrome among death patients was 100%.

Mean T3 level in death patients was 0.3ng/ml

Mean T4 level in death patients was 58ng/ml

Mean TSH level in death patients was 0.77 micro unit/ml.

THYROID HORMONE STATUS IN SICK EUTHYROID PATIENTS



THYROID HORMONE STATUS IN SICK EUTHYROID PATIENTS

	LOW T3	**LOW T3 and T4**	**LOW T3,T4.TSH**	**TOTAL**
SES+ve patients	15(71%)	5(24%)	1(5%)	21

In our study, in sick euthyroid syndrome patients most common presentation was LOW T3 (71%), second most common was LOW T3 & T4 (24%) and least common was LOW T3,T4,TSH (5%).

MEAN LEVELS OF HORMONES AND SICK EUTHYROID SYNDROME

	SES +ve pts(n)	**Std deviation**	**SES-ve pts(n)**	**Std deviation**	**TOTAL**	**Std deviation**	**p value**
T3mean (ng/ml)	0.41(21)	0.0210	1.02(29)	0.3561	0.77(50)	0.40716	0.000**
T4 mean (ng/ml)	69(21)	23.2382	87(29)	17.4245	80(50)	21.81526	0.003**
TSH mean (micro unit/ml)	1.33(21)	0.8935	1.73(29)	1.0784	1.56(50)	1.01452	0.176**

In our study, in sick euthyroid syndrome positive patients

Mean T3 level - 0.41ng/ml,

Mean T4 level - 69ng/ml,

Mean TSH level - 1.33micro unit /ml

In our study, in sick euthyroid syndrome negative patients

Mean T3 level - 1.02ng/ml,

Mean T4 level - 87ng/ml,

Mean TSH level - 1.73micro unit /ml

MEAN LEVELS OF HORMONES AND SICK EUTHYROID SYNDROME

	D1SES +ve pts(n)	**Std deviation**	**D7SES +ve pts(n)**	**Std deviation**	**p value**
T3mean (ng/ml)	0.41(18)	0.0185	0.6839(18)	.1312	<.001**
T4 mean (ng/ml)	71(18)	23.4377	84.1111(18)	16.8484	<.001**
TSH mean (micro unit/ml)	1.43(18)	0.9233	1.6061(18)	.8201	0.006**

In our study, in sick euthyroid syndrome positive patients at Day 1

Mean T3 level - 0.41ng/ml,

Mean T4 level - 71ng/ml,

Mean TSH level - 1.43micro unit /ml

In our study, in sick euthyroid positive patients repeat thyroid function test at Day7

Mean T3 level - 0.68ng/ml,

Mean T4 level - 84ng/ml,

Mean TSH level - 1.61micro unit /ml

This observation was statistically significant with above p values.

DISCUSSION

OCCURRENCE OF SICK EUTHYROID AMONG MI PATIENTS

In our study 42% of acute ST elevation MI patients had sick euthyroid syndrome. So occurrence of sick euthyroid syndrome in our patients was 42%. p value was 0.258. So occurrence of sick euthyroid positivity rate was not statistically significant. This implies that occurrence of sick euthyroid positivity rate may vary.

In Eber B et al the occurrence of sick euthyroid syndrome in patients with acute myocardial infarction has been reported was 40 %. [1]

In overall the occurrence of one or more abnormalities of thyroid function tests in patients with non thyroidal medical illnesses like starvation, sepsis, surgery, myocardial infarction, CABG surgery, bone marrow transplantation, etc …has been reported from 40% to 70%.

SEX DISTRIBUTION AND SICK EUTHYROID SYNDROME

In our study occurrence of sick euthyroid syndrome was 60% among females and 37.5% among males. This observation was statistically insignificant with the p value of 0.50. In previous studies also the occurrence of sick euthyroid syndrome in sex distribution was same.

AGE GROUP & SICK EUTHYROID SYNDROME

In our study in the age group of 20 to 39, no one had sick euthyroid syndrome. In the age group of 40 to 59, 37% had sick euthyroid syndrome. In the age group of >60, 55% of patients had sick euthyroid syndrome. p value was 0.206 that statistically insignificant. So In our study age group does not influence the occurrence of sick euthyroid syndrome positivity.

TYPES OF MI AND SICK EUTHYROID SYNDROME

In our study occurrence of sick euthyroid syndrome in AWMI patients was 36%, in IWMI patients was 42% and in others 100%. p value was 0.20 that statistically insignificant. So In our study type of MI does not influence the occurrence of sick euthyroid syndrome positivity.

To the best of our knowledge no study compared the type of MI and sick euthyroid syndrome.

KILLIP CLASS AND SICK EUTHYROID SYNDROME

In Killip T, Kimball JT et al[2] study, KILLIP CLASS predicted the severity and mortality in acute coronary syndrome. In Killip class I the mortality rate was 6%, Killip class II was 17%, Killip class III was 38% and Killip class IV was 81%.

In our study patients who presented with KILLIP 1 the sick euthyroid syndrome occurrence was 25%, in KILLIP 2 was 59%, in KILLIP 3 was 67% and KILLIP 4 was 100%.p value was 0.05* that statistically significant between the KILLIP 1 and KILLIP 2.

In our study KILLIP 2 patients had(higher mortality and morbidity) higher occurrence of sick euthyroid syndrome (59%) to KILLIP 1(25%) This observation was statistically significant with P value of .05*.

In our study 47.6% of sick euthyroid positive patients are in KILLIP 2 .But sick euthyroid negative patients only 24.1% are in KILLIP 2.p value was 0.032** that statistically significant . In Killip T, Kimball JT et al[2] study,higher the KILLIP CLASS higher the severity and mortality in acute coronary syndrome reported.

In our study Sick euthyroid syndrome positivity patients have higher KILLIP class than negative patients. Considering this Sick euthyroid syndrome positivity status predicts the severity and mortality in acute myocardial infarction.

In our study Mean T3 level, Mean T4 level, Mean TSH level progressively decreased in patients who presented with increased severity as evidenced by KILLIP class. The decrease of Mean level of T3,T4 in various class of KILLIP was statistically significant. The decrease of Mean level of TSH in various class of KILLIP was statistically insignificant. In Pavlou HN et al. Angiology 2002; 53: 699-707. Study also observed that progressive decrease of Mean T3 level, Mean T4 level and Mean TSH level with increased severity of myocardial infarction in which severity was assessed by KILLIP class. In Friberg L et al Arch Intern Med 2002; 162: 1388-1394.study observed that mortality was high among patients with the most pronounced thyroid level depression. Medha Rajappa and S.K. Sen et al[3] Study concluded that occurrence of sick euthyroid syndrome and the degree of T3 decrease is proportional to the severity of cardiac damage and may have a possible prognostic value So analyzing various studies our study also predicts the severity by more suppression of thyroid hormone.

Thus, T3 blood levels may contribute to the elaboration of an AMI severity index.

EJECTION FRACTION AND SICK EUTHYROID SYNDROME

In our study, patients presented with LVEF<50 the occurrence of sick euthyroid syndrome was 60% and patients who presented with LVEF>50 was 24%. In our study, patients who presented with LVEF<50 the sick euthyroid syndrome positivity was high compare to high LVEF >50. p value was0.01** that statistically significant.

In our study 71.4% of sick euthyroid positive patients are in LVEF<50 But 34.5% of sick euthyroid negative patients only in this group. p value was 0.010** that statistically significant. In Hallstrom A et al[7] . J Am Coll Cardiol. 1995 May;25(6):1250-7.study concluded that low left ventricular ejection fraction are strongly associated with morbidity and mortality after acute myocardial infarction . Sick euthyroid positive patients are high likelihood to have LVEF<50. So the sick euthyroid syndrome positivity predicts the high mortality and morbidity than negative individuals in acute myocardial infarction.

In our study ,patients who presented with LVEF<50 Mean T3 level ,Mean T4 level, Mean TSH level were moderately low compared to patients who presented with LVEF>50. In our study patients who presented with the LVEF <50 and LVEF>50 the decrease of MeanT3level, Mean T4 level and Mean TSH level between these groups was statistically significant.

In Medha Rajappa and S.K. Sen et al Biomedical Research 2005; 16 (1): 15-18 study observed that extent of decrease of Mean T3 level ,Mean T4 level, Mean TSH level was more significant in patients in group I (with LVEF < 50%), who have a worse prognosis than those in group II.

So analyzing various studies our study also observed when the severity was increased as evidenced by low LVEF more suppression of thyroid hormone level occurs.

ICU STAY AND SICK EUTHYROID SYNDROME

In our study in sick euthyroid positive patients mean ICU stay duration was 4.39 days compare to sick euthyroid syndrome negative patients in whom mean ICU stay duration was 3.45 days with p value of 0.007** so this observation was statistically significant. This implies that sick euthyroid positive patients have high morbidity.

RISK FACTORS AND SICK EUTHYROID SYNDROME

In our study patients who had DIABETES the occurrence of sick euthyroid syndrome was 60% and patients who presented with NO DIABETES the occurrence of sick euthyroid syndrome was 37.5% .The p value was insignificant. So in our study the presence of DIABETES does not influence the occurrence of sick euthyroid syndrome.

In our study patients who had HYPERTENSION the occurrence of sick euthyroid syndrome was 58% and patients who presented with NO HYPERTENSION the occurrence of sick euthyroid syndrome was 32%. The p value was insignificant. So in our study the presence of HYPERTENSION does not influence the occurrence of sick euthyroid syndrome.

In our study patients with risk factor SMOKING the occurrence of sick euthyroid syndrome was 41%, patients who presented with NO SMOKING the occurrence of sick euthyroid syndrome was 43%. The p value was insignificant. So in our study the presence of SMOKING habit does not influence the occurrence of sick euthyroid syndrome.

In our study patients who had ALCOHOL intake the occurrence of sick euthyroid syndrome was 39%, patients who presented with no H/O of ALCOHOL intake the occurrence of sick euthyroid syndrome was 44%. The p value was insignificant. So in our study the H/O ALCOHOL intake does not influence the occurrence of sick euthyroid syndrome.

To the best of our knowledge no study compared the risk factors and sick euthyroid syndrome.

TOTAL CHOLESTEROL LEVEL AND SICK EUTHYROID SYNDROME

In our study patients who presented with fasting cholesterol level <200 mg the occurrence of sick euthyroid syndrome was 53%, fasting cholesterol level 200 to 239mg the occurrence of sick euthyroid syndrome was 25% and fasting cholesterol level >240mg the occurrence of sick euthyroid syndrome was 47%. p value was 0.50 that statistically insignificant. So in our study cholesterol level does not influence the sick euthyroid positivity.

TRIGLYCERIDE LEVEL AND SICK EUTHYROID SYNDROME

In our study patients who presented with fasting TRIGLYCERIDE level <200 mg the occurrence of sick euthyroid syndrome was 46% and fasting TRIGLYCERIDE level >200mg the occurrence of sick euthyroid syndrome was 31%. p value was 0.50 that statistically insignificant. So in our study TRIGLYCERIDE level does not influence the sick euthyroid positivity.

HDL LEVEL AND SICK EUTHYROID SYNDROME

In our study patients who presented with fasting HDL level <40mg the occurrence of sick euthyroid syndrome was 46% and fasting HDL level >40mg the occurrence of sick euthyroid syndrome was 38%. p value was 0.50 that statistically insignificant. So in our study HDL level does not influence the sick euthyroid positivity.

DEATH AND SICK EUTHYROID SYNDROME

In our study occurrence of sick euthyroid syndrome among death patients was 100%.

Mean T3 level in death patients was 0.39ng/ml

Mean T4 level in death patients was 58ng/ml

Mean TSH level in death patients was 0.7micro unit/ml.

MEAN LEVELS OF HORMONES AND SICK EUTHYROID SYNDROME

In our study, sick euthyroid syndrome positive patients had

Mean T3 level - 0.41ng/ml,

Mean T4 level - 69ng/ml,

Mean TSH level - 1.33micro unit /ml

and sick euthyroid syndrome negative patients had

Mean T3 level - 1.02ng/ml,

Mean T4 level - 87ng/ml,

Mean TSH level - 1.73micro unit /ml.

On comparison between the groups of sick euthyroid positive and negative patients, the decrease of Mean T3,Mean T4 level in sick euthyroid positive patients was statistically significant as evidenced by p value. But the decrease of Mean TSH level in sick euthyroid positive patients was statistically insignificant.

In our study, in sick euthyroid syndrome positive patients repeat thyroid function test done at Day 7, the Mean T3 level was 0.68ng/ml, Mean T4 level was 84ng/ml and Mean TSH level was 1.61micro unit /ml.

This observation was statistically significant when we compare to Mean T3,Mean T4 and Mean TSH was done at Day 1.This observation confirms the occurrence of sick euthyroid syndrome and indicates it was transient manifestation.

LIMITATIONS OF THE STUDY

1. Thyroid function test done only at I st day and 7 th day.

2. Free T3, free T4, Reverse t3 is not measured in our study

3. Secondary hypothyroidism could not be excluded in our study patients.

4. Our study was a small study done with 50 patients only.

CONCLUSION

Occurrence of sick euthyroid syndrome was common in acute ST elevation myocardial infarction patients.

Our study shows that of 42% acute ST elevation myocardial infarction patients had sick euthyroid syndrome positivity.

The commonest abnormalities is found to be a low level of total T3 as demonstrated in our study where 71% of the patients had low level total T3.

The next most common abnormalities is found to be both low level of total T3 &total T4(24%)

As the severity of the illness increases sick euthyroid syndrome positivity rate was increased. (KILLIP 1 sick euthyroid syndrome positivity was 25%, in KILLIP 2 patients positivity was 59%, in KILLIP 3 patients was 67% and KILLIP 4 patient was 100%. patients presented with LVEF<50 the positivity of sick euthyroid syndrome was 60% and patients presented with LVEF>50 was 24%)

The degree of T3 decrease is proportional to the severity of cardiac damage and may have a possible prognostic value. (LVEF<50 Mean T3 level 0.62 ng/ml, LVEF>50 Mean T3 level 0.9ng/ml)

The changes in the thyroid hormone status return to normal once the patient recovers from the critical illness.

Though these patients have abnormalities in the thyroid hormone status they are clinically euthyroid.

Thyroid hormone system is rapidly down regulated in acute myocardial infarction. This may be beneficial during acute ischemia.

The sick euthyroid syndrome positivity rate is proportional to the severity of cardiac damage (as evidenced by KILLIP class and Ejection fraction) and may have a possible prognostic value. Thus sick euthyroid syndrome positivity may contribute to the elaboration of an AMI severity index.

The role of thyroid hormone replacement as a method of treatment of sick euthyroid syndrome is still controversial and there are no proper studies to recommend this.

Treatment of underlying condition is the treatment of choice.

AREAS OF FUTURE RESEARCH

1.Thyroid function tests including total T3,free T3,reverse T3,total T4,free T4 and TSH may be done on day 1, day4,day7and 30^{th} day and severity can be correlated with APACHE score and thyroid hormone levels.

2. Thyroid function tests including total T3, free T3, reverse T3, total T4, free T4 and TSH may be done on unstable angina and congestive cardiac failure patients.

3. To study the effect of thyroid hormone supplementation and to improve the prognosis of acute ST elevation myocardial infarction patients with Sick euthyroid syndrome positivity.

RECOMMENDATIONS

1.The presence of Sick euthyroid syndrome in acute ST elevation myocardial infarction patients may contribute to the elaboration of an AMI severity index.

SUMMARY

Euthyroid sick syndrome can be described as abnormal findings on thyroid function tests that occur in the setting of a non thyroidal illness (NTI) without preexisting hypothalamic-pituitary and thyroid gland dysfunction.

A decreased level of serum total triiodothyronine (T_3) is the most common thyroid function abnormality in patients with acute illness[14] and can be detected within 2 hours after the onset of severe physical stress.[25] As the severity of illness progresses thyroxine (T_4).[26,27] also decreased.

Acute myocardial infarction (AMI) is one of the most common critical illness in hospitalized patients.

Various studies in western world showed the occurrence of sick euthyroid syndrome in acute ST elevation myocardial infarction patients and such changes had a prognostic significance in determining severity the of AMI.

The aim of the present investigation was to confirm the occurrence of sick euthyroid syndrome in acute ST elevation myocardial infarction in our patients and to evaluate whether such changes have a prognostic significance in determining the severity of AMI.

This study was conducted in the coronary care unit, GGH, Chennai in collaboration with institute of internal medicine, institute of biochemistry and institute of Cardiology. It was a prospective study done during the period from Jan 2009 –Oct 2009 .50 patients with history, clinical features suggestive of ST elevation myocardial infarction were selected irrespective of age and sex.

Patients with clinical parameters and ECG suggestive of ST elevation myocardial infarction were taken into consideration. Each patient registered for the study went through detailed clinical evaluation as per the proforma. The severity was assessed by KILLIP classification criteria and ejection fraction. The serum of patients was analyzed for thyroid function tests at day 1 (T3,T4,TSH)and in sick euthyroid positive patients repeat thyroid function tests done at day 7 to determine the reversal of hormone status.

In our study KILLIP 2 patients (higher mortality and morbidity)had higher positivity of sick euthyroid syndrome (59%) than KILLIP 1(25%) and in patients who presented with LVEF<50 the sick euthyroid syndrome positivity was higher compare to high LVEF >50. Mean T3 level, Mean T4 level, Mean TSH level progressively decreased in patients presented with increased severity as evidenced by KILLIP class and ejection fraction and these values are statistically significant.

So compare to previous studies our study also proved that occurrence of sick euthyroid syndrome positivity and such positivity and level of progressive decrease of thyroid hormones predicts the severity of cardiac damage in our acute ST elevation myocardial infarction patients.

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OCURRENCE OF SICK EUTHYROID SYNDROME IN ACUTE ST ELEVATION MYOCARDIAL INFARCTION AND PROGNOSTIC SIGNIFICANCE.

NAME

AGE

SEX

DATE OF ADMISSION

DATE OF SAMPLING

ADMISSION DIAGNOSIS

BRIEF HISTORY

PAST HISTORY

PERSONAL HISTORY

PHYSICAL EXAMINATION

GENERAL EXAMINATION

MARKERS OF THYROID DISORDERS

VITAL SIGNS

- PULSE
- BP
- RESPIRATORY RATE
- RECTAL TEMPERATURE
- HEIGHT
- WEIGHT

CARDIOVASCULAR SYSTEM

- INSPECTION
- PALPATION
- PERCUSSION
- AUSCULTATION
- KILLIP CLASS

RESPIRATORY SYSTEM

GASTROINTESTINAL SYSTEM

CENTRAL NERVOUS SYSTEM

GCS

VENTILATORY SUPPORT

INVESTIGATIONS

CBC

- TC
- DC
- PCV
- HEMATOCRIT
- ESR
- PLATELETS

RFT

- BLOOD SUGAR
- UREA
- SERUM CREATININE
- Na+
- K+

LFT

- TOTAL BILIRUBIN
- AST
- ALT

SAP

TOTAL PROTEIN

FASTING LIPID PROFILE

ECG

RATE

RHYTHM

P WAVE

P-R INTERVAL

.QRS COMPLEX

WIDTH

AXIS

CONFIGURATION

S-T SEGMENT

T WAVE

U WAVE

COMMENTS

IMAGING

CHEST XRAY

ECHO CARDIOGRAPHY

LVEF

THYROID PROFILE DAY 1 DAY 7

TOTAL T4

TOAL T3

TSH

TREATMENT

FINAL OUTCOME

SICK – EUTHYROID STATUS :

IMPRESSION:

MASTER CHART

S.NO	AGE	SEX	DIAGNOSIS	HT	DM	SMOKING	ALCOHOL	TOTAL CHOLESTEROL	TRIGLYCERIDE	HDL
1	80	Female	IWMI	present	present	nil	nil	153	184	46
2	45	Male	IWMI	absent	absent	nil	nil	220	156	42
3	60	Male	AWMI	present	absent	yes	nil	145	138	55
4	50	Male	AWMI	absent	absent	yes	yes	270	210	39
5	50	Male	AWMI	absent	absent	yes	nil	172	118	53
6	45	Female	AWMI	absent	absent	nil	nil	234	249	46
7	55	Male	AWMI	absent	absent	yes	yes	140	61	47
8	47	Male	AWMI	absent	absent	yes	yes	140	96	50
9	57	Female	AWMI	present	absent	nil	nil	173	140	46
10	68	Male	IWMI	present	absent	yes	yes	196	104	55
11	45	Male	IWMI	absent	absent	yes	yes	162	88	49
12	43	Male	AWMI	absent	present	yes	nil	160	182	42
13	60	Female	AWMI	present	absent	nil	nil	194	160	36
14	48	Male	OTHERS	absent	absent	yes	yes	210	190	34
15	80	Male	AWMI	absent	absent	yes	yes	156	162	44
16	40	Male	AWMI	absent	absent	yes	yes	260	210	38
17	51	Male	AWMI	absent	present	nil	nil	170	130	43
18	36	Male	AWMI	absent	absent	yes	yes	242	210	38
19	67	Male	IWMI	present	present	nil	nil	286	242	33
20	45	Male	OTHERS	present	absent	yes	nil	196	210	36
21	45	Male	IWMI	absent	absent	yes	yes	230	96	43
22	65	Female	AWMI	present	absent	nil	nil	276	162	38
23	43	Male	AWMI	absent	absent	yes	yes	230	242	42
24	58	Male	IWMI	present	absent	yes	yes	224	162	32
25	55	Male	AWMI	absent	absent	nil	nil	210	120	32
26	50	Male	AWMI	present	absent	nil	nil	278	130	44
27	65	Female	OTHERS	absent	present	nil	nil	190	246	42
28	49	Female	IWMI	absent	absent	nil	nil	282	102	39
29	52	Male	AWMI	absent	absent	yes	yes	262	124	36
30	65	Male	AWMI	absent	present	nil	nil	236	210	43
31	58	Male	IWMI	absent	absent	nil	nil	260	220	32
32	60	Male	IWMI	absent	absent	nil	nil	292	178	36
33	75	Female	IWMI	present	absent	nil	nil	156	103	44
34	52	Male	IWMI	absent	absent	yes	yes	250	134	42
35	40	Male	AWMI	present	absent	yes	nil	215	150	37
36	60	Female	AWMI	absent	present	nil	nil	273	222	42
37	62	Male	AWMI	present	absent	yes	nil	290	242	39
38	82	Male	IWMI	absent	present	nil	nil	192	124	45
39	60	Male	IWMI	present	absent	yes	yes	232	130	34
40	54	Male	IWMI	present	present	nil	nil	173	192	41
41	48	Female	AWMI	present	absent	nil	nil	224	122	38
42	40	Male	AWMI	present	absent	yes	yes	210	136	34
43	46	Male	AWMI	absent	absent	yes	yes	172	190	42
44	70	Male	IWMI	absent	present	nil	yes	164	230	46
45	48	Male	AWMI	absent	absent	yes	yes	232	142	36
46	38	Male	AWMI	absent	absent	yes	yes	214	162	38
47	60	Male	AWMI	present	absent	nil	yes	262	130	34
48	41	Male	IWMI	absent	absent	yes	nil	210	174	42
49	62	Male	IWMI	present	absent	nil	yes	246	162	38
50	45	Male	IWMI	absent	absent	yes	yes	232	110	34

MASTER CHART

S.NO	KILLIP CLASS	EF	ICUSTAY	D1 T3 ng/ml	D1 T4 ng/ml	D1 TSH microunit/ml	SES	D7 T3 ng/ml	D7 T4 ng/ml	D7 TSH microunit/ml
1	2	46	5	0.40	41.00	1.80	positive	0.52	68.00	1.92
2	1	65		1.00	62.00	0.40	negative			
3	1	51	6	0.38	68.00	0.56	positive	0.58	72.00	0.63
4	2	53	2	1.20	63.00	2.20	negative			
5	1	45	5	1.35	108.00	0.47	negative			
6	1	45	3	0.78	84.00	1.28	negative			
7	2	44	4	0.40	73.00	2.80	positive	0.72	88.00	2.67
8	3	43	6	0.55	64.00	2.30	negative			
9	1	53	4	0.39	80.00	0.46	positive	0.63	85.00	0.58
10	2	44	4	0.95	77.00	0.41	negative			
11	2	43	5	0.43	41.00	0.88	positive	0.78	62.00	0.96
12	1	46	3	0.42	62.00	0.82	positive	0.64	76.00	0.99
13	1	44	5	0.52	72.00	1.20	negative			
14	3	32		0.40	70.00	1.10	positive			
15	1	34	3	1.00	75.00	0.80	negative			
16	2	56	2	1.20	85.00	0.60	negative			
17	1	46	3	1.30	95.00	0.80	negative			
18	1	58	4	1.10	90.00	2.10	negative			
19	2	40	5	0.44	62.00	0.58	positive	0.56	77.00	0.60
20	2	46	6	0.42	46.00	1.90	positive	0.92	63.00	1.80
21	1	44	4	1.40	62.00	0.40	negative			
22	2	42	4	0.46	62.00	1.80	positive	0.54	92.00	2.10
23	1	56	3	0.63	102.00	3.10	negative			
24	2	46	5	0.41	104.00	1.20	positive	0.73	109.00	1.80
25	1	50	4	0.90	98.00	3.00	negative			
26	1	53	3	1.00	70.00	3.10	negative			
27	1	56	5	0.42	102.00	4.20	positive	0.83	115.00	3.90
28	2	42	5	0.40	42.00	1.30	positive	0.67	59.00	1.60
29	4	36		0.36	32.00	0.30	positive			
30	1	52	4	1.90	102.00	3.00	negative			
31	1	60	3	0.92	122.00	4.00	negative			
32	2	56	4	1.20	96.00	2.10	negative			
33	1	58	2	0.64	70.00	3.00	negative			
34	2	52	3	0.62	92.00	2.30	negative			
35	2	46	4	0.41	68.00	2.00	positive	0.60	79.00	2.30
36	4	30		0.42	72.00	0.90	positive			
37	1	53	4	1.30	92.00	3.80	negative			
38	1	52	3	0.42	82.00	0.90	positive	0.98	87.00	1.45
39	2	43	3	0.40	102.00	0.80	positive	0.75	99.00	1.21
40	1	52	2	1.02	88.00	1.38	negative			
41	2	46	3	0.52	65.00	1.20	negative			
42	2	48	6	0.92	78.00	1.00	negative			
43	3	42	6	0.42	46.00	0.90	positive	0.62	76.00	1.30
44	1	52	3	0.82	96.00	1.80	negative			
45	1	58	2	1.20	106.00	1.10	negative			
46	1	64	4	1.90	120.00	0.60	negative			
47	1	53	3	0.41	112.00	1.40	positive	0.54	106.00	1.70
48	1	60	4	0.72	92.00	1.10	negative			
49	1	52	3	0.42	88.00	1.40	positive	0.70	101.00	1.40
50	1	56	2	0.90	110.00	1.60	negative			